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| NEWS 1 | | Web Page URLs for STN Seminar Schedule - N. America |
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| NEWS 3 | Feb 24 | PCTGEN now available on STN |
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| NEWS 13 | AUG 22 | Indexing from 1927 to 1936 added to records in CA/CAPLUS |
| NEWS 14 | Apr 21 | New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX |
| NEWS 15 | Apr 28 | RDISCLOSURE now available on STN |
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| NEWS 17 | May 15 | MEDLINE file segment of TOXCENTER reloaded |
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| NEWS 20 | May 19 | RAPRA enhanced with new search field, simultaneous left and right truncation |
| NEWS 21 | Jun 06 | Simultaneous left and right truncation added to CBNB |
| NEWS 22 | Jun 06 | PASCAL enhanced with additional data |
| NEWS 23 | Jun 20 | 2003 edition of the FSTA Thesaurus is now available |
| NEWS 24 | Jun 25 | HSDB has been reloaded |
| NEWS 25 | Jul 16 | Data from 1960-1976 added to RDISCLOSURE |
| NEWS 26 | Jul 21 | Identification of STN records implemented |
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| NEWS 28 | Jul 22 | INPADOC: Basic index (/BI) enhanced; Simultaneous Left and Right Truncation available |
| NEWS 29 | AUG 05 | New pricing for EUROPATFULL and PCTFULL effective August 1, 2003 |
| NEWS 30 | AUG 13 | Field Availability (/FA) field enhanced in BEILSTEIN |
| NEWS 31 | AUG 15 | PATDPAFULL: one FREE connect hour, per account, in September 2003 |
| NEWS 32 | AUG 15 | PCTGEN: one FREE connect hour, per account, in September 2003 |
| NEWS 33 | AUG 15 | RDISCLOSURE: one FREE connect hour, per account, in September 2003 |
| NEWS 34 | AUG 15 | TEMA: one FREE connect hour, per account, in September 2003 |
| NEWS 35 | AUG 18 | Data available for download as a PDF in RDISCLOSURE |
| NEWS 36 | AUG 18 | Simultaneous left and right truncation added to PASCAL |
| NEWS 37 | AUG 18 | FROSTI and KOSMET enhanced with Simultaneous Left and Right Truncation |

NEWS 38 AUG 18 Simultaneous left and right truncation added to ANABSTR

NEWS EXPRESS April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
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AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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NEWS WWW CAS World Wide Web Site (general information)

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* * * * * STN Columbus * * * * *

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L1 22310 (ANTIGEN) (3A) (RELEASE OR SECRETION)

=> s (kdel) (3A) (release or secretion)

36 FILES SEARCHED...

71 FILES SEARCHED...

L2 34 (KDEL) (3A) (RELEASE OR SECRETION)

=> s l1 and l2

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L3 0 L1 AND L2

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AN 2002:56228 AGRICOLA

DN IND23283389

TI Partial redistribution of the Autographa californica nucleopolyhedrovirus
chitinase in virus-infected cells accompanies mutation of the
carboxy-terminal KDEL ER-retention motif.

AU Saville, G.P.; Thomas, C.J.; Possee, R.D.; King, L.A.

AV DNAL (QR360.A1J6)

SO The Journal of general virology, Mar 2002. Vol. 83, No. pt.3. p. 685-694
Publisher: Reading : Society for General Microbiology.
CODEN: JGVIAI; ISSN: 0022-1317

NTE Includes references

CY England; United Kingdom

DT Article

FS Non-U.S. Imprint other than FAO

LA English

AB During virus infection of insect cells, the Autographa californica
nucleopolyhedrovirus chitinase is localized primarily within the
endoplasmic reticulum (ER), which is consistent with the presence of a
carboxy-terminal ER retention motif (KDEL). Release of
chitinase into the extracellular medium appears to be concomitant with
terminal cell lysis, rather than by active secretion. In this study, we
have shown that mutation of the KDEL motif induces a partial
redistribution of the chitinase at both early and late times
post-infection. Deletion of the KDEL motif or substitution with glycine
residues allowed chitinase to move through the secretory pathway,
accumulating to detectable levels in the extracellular medium by 24 h
post-infection; more than 48 h prior to cell lysis. Deletion of the KDEL
motif did not compromise enzyme activity, with the modified enzyme
exhibiting characteristic endo- and exo-chitinolytic activity.
Trichoplusia ni larvae infected with the modified virus were found to
liquefy approximately 24 h earlier than larvae infected with a control
virus in which the chitinase KDEL motif had not been deleted.

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AN 2001:334317 BIOSIS

DN PREV200100334317

TI The KDEL receptor mediates a retrieval mechanism that contributes to
quality control at the endoplasmic reticulum.

AU Yamamoto, Katsushi; Fujii, Rika; Toyofuku, Yukiko; Saito, Takashi; Koseki,
Haruhiko; Hsu, Victor W.; Aoe, Tomohiko (1)

CS (1) Department of Molecular Embryology, Chiba University Graduate School
of Medicine, Chiba, 260-8670: taoe@med.m.chiba-u.ac.jp Japan

SO EMBO (European Molecular Biology Organization) Journal, (June 15, 2001)
Vol. 20, No. 12, pp. 3082-3091. print.
ISSN: 0261-4189.

DT Article

LA English

SL English

AB Newly synthesized proteins in the endoplasmic reticulum (ER) must fold and
assemble correctly before being transported to their final cellular
destination. While some misfolded or partially assembled proteins have
been shown to exit the ER, they fail to escape the early secretory system
entirely, because they are retrieved from post-ER compartments to the ER.
We elucidate a mechanistic basis for this retrieval and characterize its
contribution to ER quality control by studying the fate of the unassembled
T-cell antigen receptor (TCR) alpha chain. While the steady-state
distribution of TCRalpha is in the ER, inhibition of retrograde transport
by COPI induces the accumulation of TCRalpha in post-ER compartments,
'suggesting that TCRalpha is cycling between the ER and post-ER

compartments. TCRalpha associates with BiP, a KDEL protein. Disruption of the ligand-binding function of the **KDEL** receptor **releases** TCRalpha from the early secretory system to the cell surface, so that TCRalpha is no longer subject to ER degradation. Thus, our findings suggest that retrieval by the KDEL receptor contributes to mechanisms by which the ER monitors newly synthesized proteins for their proper disposal.

L5 ANSWER 3 OF 9 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 1989:91574 BIOSIS
DN BA87:45710
TI EVIDENCE THAT LUMINAL ER PROTEINS ARE SORTED FROM SECRETED PROTEINS IN A POST-ER COMPARTMENT.
AU PELHAM H R B
CS MRC LAB. MOLECULAR BIOL., HILLS RD., CAMBRIDGE CB2 2QH, UK.
SO EMBO (EUR MOL BIOL ORGAN) J, (1988) 7 (4), 913-918.
CODEN: EMJODG. ISSN: 0261-4189.
FS BA; OLD
LA English
AB Several soluble proteins that reside in the lumen of the ER contain a specific C-terminal sequence (**KDEL**) which prevents their **secretion**. This sequence may be recognized by a receptor that either immobilizes the proteins in the ER, or sorts them from other proteins at a later point in the secretory pathway and returns them to their normal location. To distinguish these possibilities, I have attached an ER retention signal to the lysosomal protein cathepsin D. The oligosaccharide side chains of this protein are normally modified sequentially by two enzymes to form mannose-6-phosphate residues; these enzymes do not act in the ER, but are thought to be located in separate compartments within (or near) the Golgi apparatus. Cathepsin D bearing the ER signal accumulates within the ER, but continues to be modified by the first of the mannose-6-phosphate forming enzymes. Modification is strongly temperature-dependent, which is also a feature of ER-to-Golgi transport. These results support the idea that luminal ER proteins are continuously retrieved from a post-ER compartment, and that this compartment contains N-acetylglucosaminyl-1-phosphotransferase activity.

L5 ANSWER 4 OF 9 CANCERLIT on STN
AN 91015337 CANCERLIT
DN 91015337 PubMed ID: 2120591
TI Secretion of immunoglobulin M assembly intermediates in the presence of reducing agents.
AU Alberini C M; Bet P; Milstein C; Sitia R
CS Istituto di Chimica, Facolta di Medicina, Universita degli Studi di Brescia, Italy.
SO NATURE, (1990 Oct 4) 347 (6292) 485-7.
Journal code: 0410462. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS MEDLINE; Priority Journals
OS MEDLINE 91015337
EM 199011
ED Entered STN: 19941107
Last Updated on STN: 19970509
AB There are several demonstrations that misfolded or unassembled proteins are not transported along the secretory pathway, but are retained intracellularly, generally in the endoplasmic reticulum. For instance, B lymphocytes synthesize but do not secrete IgM, and only the polymeric form of IgM is secreted by plasma cells. The C-terminal cysteine of the mu heavy chain of secreted IgM (residue 575) is involved in the intracellular retention of unpolymerized IgM subunits. Here we report that the addition of reducing agents to the culture medium, at concentrations which do not affect cell viability, terminal glycosylation, or retention of proteins in

the endoplasmic reticulum through the **KDEL** mechanism, induces **secretion** of IgM assembly intermediates by both B and plasma cells. Free joining (J) chains, which are not normally secreted by plasma cells unless as part of IgM or IgA, are also secreted in the presence of reducing agents. We propose a role for free thiol groups in preventing the unhindered transport of proteins through the secretory pathway. Under the scheme, assembly intermediates interact through their thiol groups between themselves and/or with unknown proteins of the endoplasmic reticulum. Such interactions may be prevented by altering the intracellular redox potential or by site-directed mutagenesis of the relevant cysteine residue(s).

L5 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
 AN 2003:402183 CAPLUS
 DN 138:400516
 TI Manufacture of antibodies in plant cells as fusion proteins with elastin-like peptides
 IN Scheller, Juergen; Conrad, Udo; Leps, Michael
 PA IPK- Institut Fuer Pflanzengenetik Und Kulturpflanzen Forschung, Germany
 SO PCT Int. Appl., 62 pp.
 CODEN: PIXXD2
 DT Patent
 LA German
 FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|--------|--|--|----------|------------------|----------|
| PI | WO 2003041493 | A1 | 20030522 | WO 2002-EP12773 | 20021114 |
| | W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| | RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | |
| | DE 10155862 | A1 | 20030528 | DE 2001-10155862 | 20011114 |
| PRAI | DE 2001-10155862 | A | 20011114 | | |
| AB | A method of manufg. antibodies, esp. single-chain antibodies, in increased yields in plant cells as fusion proteins with elastin-like peptides by expression of the corresponding gene is described. Yields of antibody are greatly increased when they are manufd. as these fusion proteins compared to without them and the protein can be rapidly purified by pptn. The fusion protein may contain several repeats of the elastin-like peptide, a signal peptide to direct secretion and a KDEL peptide for retention in the endoplasmic reticulum. The gene may be expressed in seed using a a strong promoter such as the cauliflower mosaic virus 35S promoter. Yields of antibody from seed of transgenic tobacco reached 17% of total protein. | | | | |
| RE.CNT | 7 | THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT | | | |

L5 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2003 ACS on STN
 AN 2000:98760 CAPLUS
 DN 132:133894
 TI Inhibition of KDEL receptor-mediated return of heat shock protein complexes to the endoplasmic reticulum and their adjuvant use
 IN Rothman, James E.; Mayhew, Mark; Hoe, Mee H.
 PA Sloan-Kettering Institute for Cancer Research, USA
 SO PCT Int. Appl., 87 pp.
 CODEN: PIXXD2
 DT Patent

LA English

FAN.CNT 1

| | PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|----|---------------|--|----------|-----------------|----------|
| PI | WO 2000006729 | A1 | 20000210 | WO 1999-US17147 | 19990728 |
| | W: | AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | |
| | RW: | GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG | | | |
| | US 6160088 | A | 20001212 | US 1998-124671 | 19980729 |
| | CA 2337692 | AA | 20000210 | CA 1999-2337692 | 19990728 |
| | AU 9953245 | A1 | 20000221 | AU 1999-53245 | 19990728 |
| | EP 1100906 | A1 | 20010523 | EP 1999-938851 | 19990728 |
| | R: | AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO | | | |

PRAI US 1998-124671 A 19980729

WO 1999-US17147 W 19990728

AB Inhibitors of the KDEL receptor that can be used to block the transfer of heat shock proteins to the endoplasmic reticulum and allow them to act as adjuvants are described. Certain proteins are functionally retained in the cellular endoplasmic reticulum via an interaction between a KDEL sequence and its receptor. According to the invention, blocking this interaction with a KDEL receptor inhibitor promotes the secretion of such proteins. In specific embodiments of the invention, KDEL receptor inhibitors may be used to promote the secretion of heat shock proteins, thereby rendering the secreted heat shock proteins more accessible to the immune system and improving the immune response to heat shock protein-associated antigens. The inhibitors are artificial peptides that oligomerize and present large no. of KDEL peptides to the receptors and saturate them. An example of one of these peptides uses the signal peptide of the BiP protein, an oligomerization domain of a cartilage oligomeric matrix protein, a linker peptide from a camel Ig and a KDEL peptide is described.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 9 DGENE COPYRIGHT 2003 THOMSON DERWENT on STN

AN AAV41811 DNA DGENE

TI New modified pro-domain of carboxy-peptidase B - enhances expression of co-expressed proteins for production of recombinant carboxy-peptidase or its fusions with antibodies, used, e.g. in enzyme prodrug therapy

IN Edge M D

PA (ZENE) ZENECA LTD.

PI WO 9835988 A1 19980820 83p

AI WO 1998-GB415 19980210

PRAI GB 1997-22727 19971029

GB 1997-3104 19970214

GB 1997-22003 19971018

DT Patent

LA English

OS 1998-467168 [40]

DESC Human carboxypeptidase B pro-KDEL primer.

AB The pro-KDEL primer was used in the expression of human pancreatic carboxypeptidase B (CPB) from COS cells by co-secretion of pro-KDEL. The co-expression of a modified pro-domain of CPB from a separate gene enhances recombinant expression. This process can be used to produce recombinant CPB in eukaryotic cells, or fusions of CPB with antibody chains. CPB is used in insulin production and protein sequencing, while its fusions with antibody are useful in

antibody-directed enzyme prodrug therapy. The Modified pro-domain provide increased yields of recombinant CPB, possibly by protecting the C-terminus against enzymatic degradation or by increasing intracellular trafficking.

L5 ANSWER 8 OF 9 USPATFULL on STN
AN 2000:168135 USPATFULL
TI KDEL receptor inhibitors
IN Rothman, James E., New York, NY, United States
Mayhew, Mark, Tarrytown, NY, United States
Hoe, Mee H., Irvington, NY, United States
PA Sloan-Kettering Institute For Cancer, New York, NY, United States (U.S. corporation)
PI US 6160088 20001212
AI US 1998-124671 19980729 (9)
DT Utility
FS Granted
EXNAM Primary Examiner: Achutamurthy, Ponnathapu; Assistant Examiner: Tung, Peter P.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 30 Drawing Page(s)
LN.CNT 1537

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to inhibitors of the KDEL receptor and therapeutic uses therefor. Certain proteins are functionally retained in the cellular endoplasmic reticulum via an interaction between a KDEL sequence and its receptor. According to the invention, blocking this interaction with a KDEL receptor inhibitor promotes the secretion of such proteins. In specific embodiments of the invention, KDEL receptor inhibitors may be used to promote the secretion of heat shock proteins, thereby rendering the secreted heat shock proteins more accessible to the immune system and improving the immune response to heat shock protein-associated antigens.

L5 ANSWER 9 OF 9 USPATFULL on STN
AN 1999:141293 USPATFULL
TI Recombinant vectors for reconstitution of liver
IN Kay, Mark A., Seattle, WA, United States
Lieber, Andre, Seattle, WA, United States
PA University of Washington, Seattle, WA, United States (U.S. corporation)
PI US 5980886 19991109
AI US 1997-819377 19970317 (8)
RLI Continuation of Ser. No. US 1995-476257, filed on 7 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-357508, filed on 14 Dec 1994, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Stanton, Brian R.; Assistant Examiner: Clark, Deborah J. R.
LREP Campbell & Flores
CLMN Number of Claims: 13
ECL Exemplary Claim: 1,8
DRWN 18 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1301

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A combination of retroviral and adenoviral vectors are used for high efficiency gene transfer into hepatocytes, resulting in long term gene expression. Hepatocytes are transduced in vivo with a recombinant adenovirus vector that expresses a molecule capable of inducing hepatocyte regeneration, such as urokinase plasminogen activator (uPA) or tissue plasminogen activator (tPA), resulting in a high rate of liver regeneration. During the regenerative phase, ex vivo or in vivo retroviral-mediated gene transfer into hepatocytes results in greater

transduction efficiencies. The compositions and methods thus provide new means for gene therapy, and transgenic non-human animals useful in developing new therapeutic and preventative agents.

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<-----User Break----->

=> s (antigen) (3A) (release or secretion or released or secreted or release or secrete)

19 FILES SEARCHED...

33 FILES SEARCHED...

51 FILES SEARCHED...

66 FILES SEARCHED...

L6 35952 (ANTIGEN) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED OR
RELEASE OR SECRETE)

=> (kdel) (3A) (release or secretion or released or secreted or release or secrete)
(KDEL) IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.

For a list of commands available to you in the current file, enter

"HELP COMMANDS" at an arrow prompt (=>).

=> s (kdel) (3A) (release or secretion or released or secreted or secrete)

27 FILES SEARCHED...

54 FILES SEARCHED...

84 FILES SEARCHED...

L7 58 (KDEL) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED OR
SECRETE)

=> s l6 and l7

63 FILES SEARCHED...

75% OF LIMIT FOR L#S REACHED

93 FILES SEARCHED...

L8 1 L6 AND L7

=> d l8 bib ab

L8 ANSWER 1 OF 1 USPATFULL on STN

AN 1999:40194 USPATFULL

TI Method of producing single-chain Fv molecules

IN Jost, Carolina R., Washington, DC, United States

Segal, David M., Rockville, MD, United States

Huston, James S., Chestnut Hill, MA, United States

PA The United States of America as represented by the Department of Health
and Human Services, Washington, DC, United States (U.S. government)

PI US 5888773 19990330

AI US 1994-292124 19940817 (8)

DT Utility

FS Granted

EXNAM Primary Examiner: Huff, Sheela; Assistant Examiner: Reeves, Julie E.

LREP Townsend and Townsend and Crew LLP

CLMN Number of Claims: 14

ECL Exemplary Claim: 1

DRWN 12 Drawing Figure(s); 12 Drawing Page(s)

LN.CNT 1407

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a method of producing single-chain Fv molecules
in eukaryotic cells, and to secretable sFv proteins having at least one
non-naturally occurring glycosylation site. The single-chain Fv
molecules produced by this method are biologically active and capable of
being secreted from eukaryotic cells into the cell culture medium.

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=> s l6 and (kdel)
    38 FILES SEARCHED...
    94 FILES SEARCHED...
L9          26 L6 AND (KDEL)
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=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l9
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FEDRIP, FOREGE, GENBANK, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, RDISCLOSURE, SYNTHLINE, CHEMLIST,
HSDB, MSDS-CCOHS, MSDS-OHS, RTECS, CONF, IMSDRUGCONF, DIOGENES, INVESTEXT,
USAN, FORIS, FORKAT, UFOR DAT, AQUIRE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
DUPLICATE PREFERENCE IS 'BIOTECHABS, CAPLUS, USPATFULL, WPINDEX'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L9
L10          25 DUPLICATE REMOVE L9 (1 DUPLICATE REMOVED)
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=> s l10 NOT (genebank or genbank)
    22 FILES SEARCHED...
    95% OF LIMIT FOR L#S REACHED
    42 FILES SEARCHED...
    58 FILES SEARCHED...
MAXIMUM L# REACHED
SAVE QUERIES AND/OR ANSWER SETS IF DESIRED
USE DELETE HISTORY TO DELETE ALL L'S
MAXIMUM L# WOULD BE EXCEEDED
SAVE QUERIES AND/OR ANSWER SETS IF DESIRED
USE DELETE HISTORY TO DELETE ALL L#S
If this command were executed now, it would create an L-number higher
than the maximum allowed L-number (L999). To perform this command,
you must delete some or all L-numbers. SAVE any L-number queries or
answer sets you wish to retain, then execute DELETE HISTORY.
```

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=> delete history
DELETE ALL L# ITEMS? (Y)/N:y
ALL L# ITEMS DELETED

=> s ((antigen) (3A) (release or secretion or released or secreted or release or
secrete)) and (kdel)
  19 FILES SEARCHED...
  34 FILES SEARCHED...
  54 FILES SEARCHED...
  72 FILES SEARCHED...
L1      26 ((ANTIGEN) (3A) (RELEASE OR SECRETION OR RELEASED OR SECRETED
OR RELEASE OR SECRETE)) AND (KDEL)

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l1
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE,
DRUGLAUNCH, DRUGMONOG2, DRUGUPDATES, FEDRIP, FOREGE, GENBANK, KOSMET,
MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, RDISCLOSURE, SYNTHLINE, CHEMLIST,
HSDB, MSDS-CCOHS, MSDS-OHS, RTECS, CONF, IMSDRUGCONF, DIOGENES, INVESTEXT,
USAN, FORIS, FORKAT, UFORDAT, AQUIRE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
DUPLICATE PREFERENCE IS 'BIOTECHABS, CAPLUS, USPATFULL, WPINDEX'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L1
L2      25 DUPLICATE REMOVE L1 (1 DUPLICATE REMOVED)

=> s l2 NOT (genbank) or genebank)
UNMATCHED RIGHT PARENTHESIS 'GENEBANK)'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s l2 NOT (genbank or genebank)
  29 FILES SEARCHED...
  45 FILES SEARCHED...
  58 FILES SEARCHED...
  92 FILES SEARCHED...
L3      17 L2 NOT (GENBANK OR GENEBAK)

=> d l3 1-17 bib ab

L3      ANSWER 1 OF 17 BIOTECHABS COPYRIGHT 2003 THOMSON DERWENT/ISI on STN
AN      2002-17633 BIOTECHABS
TI      Preparing recombinant vector containing reporter and therapeutic genes,
        useful for treatment of fibrosis, particularly of liver, by inducing
        degradation of collagen;
        recombinant adeno virus production expressing beta-galactosidase,
        human urokinase, transforming growth factor and growth factor gene
AU      ARMENDARIZ BORUNDA J; AGUILAR CORDOVA E
PA      TGT LAB SA DE CV
PI      WO 2002044393 6 Jun 2002
AI      WO 2000-MX50 28 Nov 2000
PRAI    MX 2000-1713 28 Nov 2000
DT      Patent
LA      Spanish
OS      WPI: 2002-471834 [50]
AB      DERWENT ABSTRACT:
        NOVELTY - Preparing recombinant (non-)viral vectors (A) by cloning
        reporter gene (RG), and modified cDNA (I) of a therapeutic gene (II) that
        encodes a protein (III) useful for treating fibrosis (hepatic, pulmonary,
        renal, cardiac or pancreatic), keloids and hypertrophic scars in mammals,
        including humans, is new.
        DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a
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recombinant adenoviral vector (A') produced by the new method.

BIOTECHNOLOGY - Preferred Materials: RG is a bacterial beta-galactosidase (lacZ) gene. (III) is: (a) optionally modified human urokinase-type plasminogen activator (huPA) (this activates latent hepatic collagenases and matrix metalloproteases, and restores replication of hepatocytes); (b) truncated type II TGF (transforming growth factor) beta receptor (this degrades and/or prevents synthesis/deposition of collagen proteins in cirrhotic organs); or (c) hepatic growth factor (this induces hepatic regeneration, leading to an increase in the number of hepatocytes positive for cellular proliferation nuclear antigen). A non-secreted form of huPA is expressed, i.e. one that has a signal for retention in the endoplasmic reticulum (ER), specifically KDEL, at the C-terminus, also, at the N-terminus, a sequence that encodes a retention signal linked to a transmembrane region anchor. Preferred Vectors: The vectors are adenoviral (1st-4th generation or 'gutless'), retroviral or adeno-associated viral vectors, or plasmids or cationic and anionic liposomes. Especially, preferred is a recombinant adenoviral vector in which the E1 and/or E3 open reading frames have been removed (but leaving sufficient sequence for vector replication in vitro) and the inserted gene or cDNA is under control of an ubiquitous, tissue-specific and/or inducible or regulatable promoter, especially the cytomegalovirus promoter. Preferred Process: Expression of the therapeutic gene can be monitored by measuring expression of the corresponding human protein (enzyme-linked immunosorbent assay or immunohistochemical methods), and expression of endogenous collagen genes may be followed by semi-quantitative reverse-transcription polymerase chain reaction.

ACTIVITY - Hepatotropic; Antifibrotic; Vulnerary.

MECHANISM OF ACTION - (II) induce degradation of collagen. Liver cirrhosis was induced in rats using tetrachloromethane, then the animals injected (iliac vein) with a single dose of 6×10^{11} viral particles/kg of vector pAd.PGK-DELTANDELTAAC-huPA (expressing human urokinase). Analysis of the liver after 8 days indicated levels (international units/l) of ALT, AST and alkaline phosphatase of 88.5, 137 and 205; compare 410, 2250 and 454 for animals injected with an irrelevant vector; and total bilirubin was 0.94 (compare 1.4) mg/dl. Hematological parameters were not affected and the treatment significantly increased expression of matrix metalloprotease-2; reduced expression of collagens types I, III and IV, and stimulated mitosis of hepatocytes.

USE - (A) are used to treat fibrosis in the cirrhotic liver, but more generally fibrosis in any organ.

ADMINISTRATION - Targeting to fibrotic organs is achieved by selecting the route of administration (specifically intravenous); from the natural tropism (to liver) of the vector, or by vector selection. The unit dose is 10^7 - 10^{14} viral particles.

ADVANTAGE - (A) do not secrete significant amounts of plasminogen activator, so cause neither hypocoagulation nor spontaneous bleeding.

EXAMPLE - The cDNA for human urokinase plasminogen activator (huPA) was cloned into the XbaI/Asp718 sites of pGEM3 and modified by attachment of a sequence encoding the signal KDEL, for retention in the endoplasmic reticulum, at the C-terminus and a 75 base pair polymerase chain reaction fragment to replace the pre-uPA signal sequence with a retention signal and transmembrane anchor from the transmembrane protein IiP33. The modified cDNA was then cloned (no details) to form vector pAdPGK-DELTANDELTAAC-huPA, for subsequent production of recombinant adenoviral particles. (74 pages)

L3 ANSWER 2 OF 17 CAPLUS COPYRIGHT 2003 ACS on STN

AN 1993:58075 CAPLUS

DN 118:58075

TI The calcium-binding protein calreticulin is a major constituent of lytic granules in cytolytic T lymphocytes

AU Dupuis, Marc; Schaerer, Esther; Krause, Karl Heinz; Tschopp, Juerg

CS Inst. Biochem., Univ. Lausanne, Epalinges, CH-1066, Switz.
SO Journal of Experimental Medicine (1993), 177(1), 1-7
CODEN: JEMEA; ISSN: 0022-1007
DT Journal
LA English
AB Cytolytic T lymphocytes (CTL), natural killer cells, and lymphokine-activated killer (LAK) cells are cytolytic cells known to release the cytolytic protein perforin and a family of proteases, named granzymes, from cytoplasmic stores upon interaction with target cells. Here, the authors report the purifn. of an addnl. major 60-kD granule-assocd. protein (grp 60) from human LAK cells and from mouse cytolytic T cells. The N-terminal amino acid sequence of the polypeptide was found to be identical to calreticulin. Calreticulin is a Ca storage protein and carries a C-terminal KDEL sequence, known to act as a retention signal for proteins destined to the lumen of the endoplasmic reticulum. In CTLs, however, calreticulin colocalizes with the lytic perforin to the lysosome-like secretory granules, as confirmed by double label immunofluorescence confocal microscopy. Moreover, when the release of granule-assocd. proteins was triggered by stimulation of the T cell receptor complex, calreticulin was released along with granzymes A and D. Since perforin is activated and becomes lytic in the presence of Ca, it is proposed that the role of calreticulin is to prevent organelle autolysis due to the protein's Ca chelator capacity.

L3 ANSWER 3 OF 17 USPATFULL on STN
AN 2003:232060 USPATFULL
TI Vaccine adjuvant
IN Minion, F. Chris, Ames, IA, UNITED STATES
Menon, Sreekumar A., Philadelphia, PA, UNITED STATES
Mahairas, Gregory G., Seattle, WA, UNITED STATES
PA Iowa State University Research Foundation, Inc., an Iowa corporation (U.S. corporation)
PI US 2003162260 A1 20030828
AI US 2003-384948 A1 20030310 (10)
RLI Division of Ser. No. US 2000-692064, filed on 19 Oct 2000, GRANTED, Pat. No. US 6537552
PRAI US 1999-160249P 19991019 (60)
DT Utility
FS APPLICATION
LREP FISH & RICHARDSON P.C., 3300 DAIN RAUSCHER PLAZA, 60 SOUTH SIXTH STREET, MINNEAPOLIS, MN, 55402
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 1632
AB The invention features fusion agents such as fusion proteins that are useful for the treatment of and prevention from diseases that are susceptible to the effects of cellular (Th1 type) immune responses. Also encompassed by the invention are nucleic acids encoding the fusion proteins of the invention, vectors containing the nucleic acids, and cells containing the vectors. The invention includes methods of making and using the fusion agents of the invention.

L3 ANSWER 4 OF 17 USPATFULL on STN
AN 2003:165491 USPATFULL
TI Hybrid lt-a/ct-b holotoxin for use as an adjuvant
IN Clements, John D, New Orleans, LA, UNITED STATES
PI US 2003113345 A1 20030619
AI US 2002-276844 A1 20021119 (10)
WO 2001-US16542 20010521
DT Utility
FS APPLICATION
LREP PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711
CLMN Number of Claims: 22

ECL Exemplary Claim: 1

DRWN 6 Drawing Page(s)

LN.CNT 1259

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a novel composition which is a hybrid heat labile enterotoxin comprising the A-subunit of the heat labile toxin of Escherichia coli (LT-A) and the B-subunit of the cholera enterotoxin of Vibrio cholerae (CT-B). The hybrid toxin is designated LT-A/CT-B. The LT-A subunit, the CT-B subunit, or both subunits of the hybrid toxin may be mutant subunits, e.g., differing from wild-type subunits by amino acid substitutions, deletions or additions. Also provided are methods of using the novel LT-A/CT-B comprising compositions of the invention as adjuvants for vaccines, methods of making the LT-A/CT-B hybrid holotoxin, and kits.

L3 ANSWER 5 OF 17 USPATFULL on STN

AN 2003:81455 USPATFULL

TI Vaccine adjuvant

IN Minion, F. Chris, Ames, IA, United States

Menon, Sreekumar A., Philadelphia, PA, United States

Mahairas, Gregory G., Seattle, WA, United States

PA Iowa State University Research Foundation, Ames, IA, United States (U.S. corporation)

PI US 6537552 B1 20030325

AI US 2000-692064 20001019 (9)

PRAI US 1999-160429P 19991019 (60)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Smith, Lynette R. F.; Assistant Examiner: Shahnan-Shah, Khatol S

LREP Fish & Richardson P.C.

CLMN Number of Claims: 8

ECL Exemplary Claim: 1

DRWN 10 Drawing Figure(s); 7 Drawing Page(s)

LN.CNT 1611

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention features fusion agents such as fusion proteins that are useful for the treatment of and prevention from diseases that are susceptible to the effects of cellular (Th1 type) immune responses. Also encompassed by the invention are nucleic acids encoding the fusion proteins of the invention, vectors containing the nucleic acids, and cells containing the vectors. The invention includes methods of making and using the fusion agents of the invention.

L3 ANSWER 6 OF 17 USPATFULL on STN

AN 2003:40768 USPATFULL

TI Pharmaceuticals for modulating hormone responsiveness

IN Dedhar, Shoukat, #1-219 East 8th St., North Vancouver B.C., CANADA V7L 1Y9

PI US 6518397 B1 20030211

AI US 1997-900241 19970724 (8)

RLI Continuation-in-part of Ser. No. US 377432, now patented, Pat. No. US 5854202

DT Utility

FS GRANTED

EXNAM Primary Examiner: Eyler, Yvonne; Assistant Examiner: Brannock, Michael T.

LREP Burns, Doane, Swecker & Mathis, L.L.P.

CLMN Number of Claims: 4

ECL Exemplary Claim: 1

DRWN 12 Drawing Figure(s); 11 Drawing Page(s)

LN.CNT 1930

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to isolated and purified proteins, such as

calreticulin and mimetics and inhibitors of calreticulin, for a novel use of modulating hormone responsiveness. These proteins are useful in gene therapy and in manufacturing pharmaceuticals for treating a variety of diseases, including cancer, osteoporosis and chronic inflammatory disease. The proteins include or bind to an amino acid sequence [SEQ ID NO: 1] KXFFX.sup.1R, wherein X is either G, A or V and Y is either K or R. This sequence is present in the DNA-binding domain, and is critical for the DNA binding activity, of a variety of hormone receptors, including glucocorticoid receptor, mineralocorticoid receptor, androgen receptor, progesterone receptor, estrogen receptor, retinoic acid receptor, thyroid hormone receptor and vitamin D receptor. Proteins which bind to this sequence may inhibit hormone receptor induced gene transcription. Proteins which include this sequence may promote hormone receptor induced gene transcription.

L3 ANSWER 7 OF 17 USPATFULL on STN
AN 2002:258433 USPATFULL
TI Anti-CD3 immunotoxins and therapeutic uses therefor
IN Digan, Mary Ellen, Morristown, NJ, UNITED STATES
Lake, Philip, Morris Plains, NJ, UNITED STATES
Wright, Richard Michael, Annandale, NJ, UNITED STATES
PI US 2002142000 A1 20021003
AI US 2000-480236 A1 20000110 (9)
DT Utility
FS APPLICATION
LREP THOMAS HOXIE, NOVARTIS CORPORATION, PATENT AND TRADEMARK DEPT, 564
MORRIS AVENUE, SUMMIT, NJ, 079011027
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 23 Drawing Page(s)
LN.CNT 2935
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Recombinant immunotoxin polypeptides are described comprising a CD3-binding domain and a Pseudomonas exotoxin mutant, and in particular, comprising a single chain (sc) Fv as the CD3-binding moiety. A preferred species of the invention comprises scFv(UCHT-1)-PE38. Also disclosed are methods for the preparation of said immunotoxins; functionally equivalent immunotoxins which are intermediates in the preparation of the immunotoxins of the invention, as well as polynucleotide and oligonucleotide intermediates; methods for the prevention and/or treatment of transplant rejection and induction of tolerance, as well as treatment of autoimmune and other immune disorders, using the immunotoxins or pharmaceutically acceptable salts thereof; and pharmaceutical compositions comprising the immunotoxins or pharmaceutically acceptable salts thereof.

L3 ANSWER 8 OF 17 USPATFULL on STN
AN 2002:198276 USPATFULL
TI IMPROVEMENTS IN OR RELATING TO PEPTIDE DELIVERY
IN CARDY, DONALD LEONARD NICHOLAS, NORTHAMPTONSHIRE, UNITED KINGDOM
CARR, FRANK JOSEPH, BALMEDIE, UNITED KINGDOM
PI US 2002106370 A1 20020808
AI US 1997-737457 A1 19970312 (8)
WO 1995-GB1107 19950515
PRAI GB 1994-9643 19940513
GB 1994-17461 19940831
DT Utility
FS APPLICATION
LREP ORRIN M HAUGEN, HAUGEN AND NIKOLAI, 820 INTERNATIONAL CENTRE, 900 SECOND
AVENUE SOUTH, MINNEAPOLIS, MN, 554023325
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 751

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed is a chimaeric polypeptide comprising: a binding portion having specific binding affinity for a eukaryotic target cell surface component and an effector portion comprising an amino acid sequence capable of exerting a biological effect; whereby binding of the polypeptide to the cell surface component induces internalization of at least the effector portion so as to allow the amino acid sequence to exert its biological effect, together with a vaccine comprising the chimaeric polypeptide of the invention, and a method of modulating the immune response of a human or animal subject.

L3 ANSWER 9 OF 17 USPATFULL on STN

AN 2002:188125 USPATFULL

TI Protease-activatable pseudomonas exotoxin A-like proproteins

IN Fitzgerald, David J., Rockville, MD, United States

Reiter, Yoram, Ness Ziona, ISRAEL

Pastan, Ira, Potomac, MD, United States

PA The United States of America as represented by the Secretary of the Department of Health and Human Services, Washington, DC, United States (U.S. government)

PI US 6426075 B1 20020730

WO 9820135 19980514

AI US 1999-297851 19990730 (9)

WO 1997-US20207 19971105

19990730 PCT 371 date

PRAI US 1996-30376P 19961106 (60)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Navarro, Mark; Assistant Examiner: Baskar, Padma

LREP Townsend and Townsend and Crew, LLP

CLMN Number of Claims: 19

ECL Exemplary Claim: 1

DRWN 9 Drawing Figure(s); 11 Drawing Page(s)

LN.CNT 2738

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides protease-activatable Pseudomonas exotoxin A-like ("PE-like") proproteins. The proproteins comprise (1) a cell recognition domain of between 10 and 1500 amino acids that binds to a cell surface receptor; (2) a modified PE translocation domain comprising an amino acid sequence sufficiently homologous to domain II of PE to effect translocation to a cell cytosol upon proteolytic cleavage, wherein the translocation domain comprises a cysteine-cysteine loop that comprises a protease activatable sequence cleavable by a protease and wherein the cysteine-cysteine loop is substantially un-activatable by furin; (3) optionally, a PE Ib-like domain comprising an amino acid sequence up to 1500 amino acids; (4) a cytotoxicity domain comprising an amino acid sequence substantially homologous to domain III of PE, the cytotoxicity domain having ADP-ribosylating activity; and (5) an endoplasmic reticulum ("ER") retention sequence. The invention also provides methods of using these proproteins for killing target cells.

L3 ANSWER 10 OF 17 USPATFULL on STN

AN 2002:181537 USPATFULL

TI Polynucleotides encoding protease-activatable pseudomonas exotoxin a-like proproteins

IN Fitzgerald, David J., Rockville, MD, United States

Reiter, Yoram, Ness Ziona, ISRAEL

Pastan, Ira, Potomac, MD, United States

PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

PI US 6423513 B1 20020723

AI US 2000-479479 20000110 (9)

RLI Division of Ser. No. US 297851

PRAI US 1996-30376P 19961106 (60)

DT Utility
FS GRANTED
EXNAM Primary Examiner: Navarro, Mark; Assistant Examiner: Baskar, Padmavathi
LREP Townsend and Townsend and Crew, LLP
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 11 Drawing Figure(s); 11 Drawing Page(s)
LN.CNT 2665

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides protease-activatable Pseudomonas exotoxin A-like ("PE-like") proproteins. The proproteins comprise (1) a cell recognition domain of between 10 and 1500 amino acids that binds to a cell surface receptor; (2) a modified PE translocation domain comprising an amino acid sequence sufficiently homologous to domain II of PE to effect translocation to a cell cytosol upon proteolytic cleavage, wherein the translocation domain comprises a cysteine-cysteine loop that comprises a protease activatable sequence cleavable by a protease and wherein the cysteine-cysteine loop is substantially un-activatable by furin; (3) optionally, a PE Ib-like domain comprising an amino acid sequence up to 1500 amino acids; (4) a cytotoxicity domain comprising an amino acid sequence substantially homologous to domain m of PE, the cytotoxicity domain having ADP-ribosylating activity; and (5) an endoplasmic reticulum ("ER") retention sequence. The invention also provides methods of using these proproteins for killing target cells.

L3 ANSWER 11 OF 17 USPATFULL on STN

AN 2002:1088 USPATFULL

TI Recombinant Haemophilus influenzae adhesin proteins

IN Loosmore, Sheena M., Aurora, CANADA

Yang, Yan Ping, Willowdale, CANADA

Klein, Michel H., Willowdale, CANADA

PA Aventis Pasteur Limited, Toronto, CANADA (non-U.S. corporation)

PI US 6335182 B1 20020101

AI US 1999-268347 19990316 (9)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Graser, Jennifer E.

LREP Sim & McBurney

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN 206 Drawing Figure(s); 201 Drawing Page(s)

LN.CNT 2173

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Recombinant production of Hia protein, in full-length and N-terminally truncated forms, of non-typeable strains of Haemophilus influenzae, is described. The nucleic acid and deduced amino acid sequences of Hia genes of various strains of non-typeable and type c Haemophilus influenzae also are described.

L3 ANSWER 12 OF 17 USPATFULL on STN

AN 2001:93332 USPATFULL

TI Immunization with plasmid encoding immunogenic proteins and intracellular targeting sequences

IN Williams, William V., Havertown, PA, United States

Madaio, Michael, Bryn Mawr, PA, United States

Weiner, David B., Merion Station, PA, United States

PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)

PI US 6248565 B1 20010619

AI US 2000-496301 20000202 (9)

RLI Continuation of Ser. No. US 1997-957001, filed on 23 Oct 1997

PRAI US 1996-29592P 19961023 (60)

DT Utility

FS GRANTED

EXNAM Primary Examiner: Park, Hankyel T.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 35
ECL Exemplary Claim: 1
DRWN 22 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1952

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Improved vaccines are disclosed. The improved vaccines include a nucleotide sequence that encodes a coding sequence that comprises an immunogenic target protein linked to or comprising an intracellular cellular targeting sequence, the coding sequence being operably linked to regulatory elements are disclosed. Methods of immunizing individuals are disclosed.

L3 ANSWER 13 OF 17 USPATFULL on STN

AN 2001:67432 USPATFULL

TI Plasmids encoding immunogenic proteins and intracellular targeting sequences

IN Williams, William V., Havertown, PA, United States

Madaio, Michael, Bryn Mawr, PA, United States

Weiner, David B., Merion Station, PA, United States

PA The Trustees of the University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)

PI US 6228621 B1 20010508

AI US 1997-957001 19971023 (8)

PRAI US 1996-29592P 19961023 (60)

DT Utility

FS Granted

EXNAM Primary Examiner: Park, Hankyel

LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP

CLMN Number of Claims: 40

ECL Exemplary Claim: 1

DRWN 22 Drawing Figure(s); 14 Drawing Page(s)

LN.CNT 1897

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Improved vaccines are disclosed. The improved vaccines include a nucleotide sequence that encodes a coding sequence that comprises an immunogenic target protein linked to or comprising an intracellular cellular targeting sequence, the coding sequence being operably linked to regulatory elements are disclosed. Methods of immunizing individuals are disclosed.

L3 ANSWER 14 OF 17 USPATFULL on STN

AN 1999:40194 USPATFULL

TI Method of producing single-chain Fv molecules

IN Jost, Carolina R., Washington, DC, United States

Segal, David M., Rockville, MD, United States

Huston, James S., Chestnut Hill, MA, United States

PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

PI US 5888773 19990330

AI US 1994-292124 19940817 (8)

DT Utility

FS Granted

EXNAM Primary Examiner: Huff, Sheela; Assistant Examiner: Reeves, Julie E.

LREP Townsend and Townsend and Crew LLP

CLMN Number of Claims: 14

ECL Exemplary Claim: 1

DRWN 12 Drawing Figure(s); 12 Drawing Page(s)

LN.CNT 1407

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a method of producing single-chain Fv molecules in eukaryotic cells, and to secretable sFv proteins having at least one non-naturally occurring glycosylation site. The single-chain Fv

molecules produced by this method are biologically active and capable of being secreted from eukaryotic cells into the cell culture medium.

L3 ANSWER 15 OF 17 USPATFULL on STN
AN 1998:161992 USPATFULL
TI Genetically engineered chimeric viruses for the treatment of diseases associated with viral transactivators
IN Tattersall, Peter J., Guilford, CT, United States
Cotmore, Susan F., Guilford, CT, United States
PA Yale University, New Haven, CT, United States (U.S. corporation)
PI US 5853716 19981229
AI US 1996-690174 19960725 (8)
PRAI US 1995-1611P 19950728 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Ketter, James; Assistant Examiner: Yucel, Irem
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 2322
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to chimeric viruses, the replication of which is regulated by a transactivation signal produced by diseased host cells. The chimeric viruses of the invention can infect both normal and diseased host cells. However, the chimeric virus replicates efficiently in and kills diseased host cells that produce the transactivation signal. The use of such chimeric viruses to treat infectious diseases and cancers are described. A particularly useful embodiment involves the modification of a murine parvovirus that infects human T cells to generate a chimeric parvovirus that is cytotoxic to human T cells that express HIV-tat. The chimeric parvovirus can be used to treat HIV-infection.

L3 ANSWER 16 OF 17 USPATFULL on STN
AN 96:11071 USPATFULL
TI Monoclonal antibodies to prostate cells
IN Pastan, Ira H., Potomac, MD, United States
PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5489525 19960206
AI US 1992-958140 19921008 (7)
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Toni R.
LREP Townsend and Townsend Kourie and Crew
CLMN Number of Claims: 11
ECL Exemplary Claim: 7
DRWN 13 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1450
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Monoclonal antibodies are provided which bind to an antigen associated with prostate cells, including prostate cancers. The monoclonal antibodies and recombinant forms thereof are used individually or conjugated radioisotopes to target the compounds to cancerous prostate cells, and thus are useful in a variety of diagnostic procedures.

L3 ANSWER 17 OF 17 WPINDEX COPYRIGHT 2003 THOMSON DERWENT on STN
AN 1999-562102 [47] WPINDEX
DNC C1999-163981
TI New polynucleotides encoding antigens which are presented with MHC Class I and II molecules, used for treating e.g. tumors, infections, autoimmune disorders, allergies or allograft rejection.
DC B04 D16

IN ROBERTS, B L
PA (GENZ) GENZYME CORP
CYC 23

PI WO 9947641 A1 19990923 (199947)* EN 82p
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP US

AU 9931022 A 19991011 (200008)

EP 1064354 A1 20010103 (200102) EN

R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

JP 2002506633 W 20020305 (200220) 94p

ADT WO 9947641 A1 WO 1999-US6030 19990319; AU 9931022 A AU 1999-31022
19990319; EP 1064354 A1 EP 1999-912709 19990319, WO 1999-US6030 19990319;
JP 2002506633 W WO 1999-US6030 19990319, JP 2000-536824 19990319

FDT AU 9931022 A Based on WO 9947641; EP 1064354 A1 Based on WO 9947641; JP
2002506633 W Based on WO 9947641

PRAI US 1998-78725P 19980320

AB WO 9947641 A UPAB: 19991116

NOVELTY - A novel polynucleotide (PN), referred to as (A), encodes an antigen that is processed and presented with a major histocompatibility complex (MHC) Class I molecule on an antigen-presenting cell (APC) and an antigen that is processed and presented with an MHC Class II molecule on the APC.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) a gene delivery vehicle comprising a PN as in (A);
- (2) a host cell comprising a PN as in (A);
- (3) a polypeptide encoded by a PN as in (A);
- (4) a method of expressing (A) by culturing the cell of (2) under standard culture conditions;
- (5) a method of increasing presentation of a peptide on the surface of an APC comprises introducing (A) into the cell under conditions which favor the expression of the polynucleotide;
- (6) a cell produced by the method of (5);
- (7) a method of producing a population of educated, antigen-specific immune effector cells comprises culturing naive immune effector cells with an APC transduced with (A);
- (8) a population of educated, antigen-specific immune effector cells produced by the method of (7);
- (9) a method of inducing an immune response to an antigen in a subject comprises administering (A) under conditions that induce an immune response to the antigen;
- (10) a method of inducing an immune response to a native antigen in a subject comprises administering the host cell of (2) under conditions that induce an immune response to the antigen; and
- (11) a method of adoptive immunotherapy comprises administering to an individual an effective amount of the cells of (8).

ACTIVITY - Immunosuppressive; Antiallergic.

MECHANISM OF ACTION - None given.

USE - The PNs can use both MHC Class I and Class II presenting pathways, in the same APC, to modulate a humoral and a cellular immune response in a subject against a given antigen. The APCs transduced with the PNs can be used for producing a population of educated, antigen-specific immune effector cells (claimed). The PNs can be used for inducing an immune response to an antigen in a subject (claimed). The antigen may be a tumor-associated antigen, e.g. gp100, MUC-1, MART-1, HER-2, CEA, PSA, prostate specific membrane antigen, tyrosinase, tyrosinase related proteins 1 or 2, NY-ESO-1 or GA733 antigen (claimed). The PNs are useful in methods in induce, increase, or enhance an immune response to a pathogenic organism, e.g. pathogenic viruses, bacteria or protozoans. They can also be used to treat disorders such as autoimmune disorders, allergies, or allograft rejection. The PNs can also be used in diagnostic applications.

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